The Effects of Obesity on Spine Surgery: A Systematic Review of the Literature

Keith L. Jackson II[1] John G. Devine[2]

[1] Department of Orthopaedics and Rehabilitation, Womack Army Medical Center, Fort Bragg, North Carolina, United States
[2] Department of Orthopaedic Surgery, Georgia Regents University, Augusta, Georgia, United States

Address for correspondence Keith L. Jackson II, MD, Staff Spine Surgeon, Department of Orthopaedics and Rehabilitation, Womack Army Medical Center, 2817 Reilly Road, Fort Bragg, NC 28310, United States (e-mail: Lynn.Jackson9@gmail.com).

Global Spine J 2016;6:394–400.

Abstract

Study Design Literature review.

Objective The aim of this literature review is to examine the effects of obesity on postoperative complications and functional outcomes after spine surgery.

Methods A review of the relevant literature examining the effects of obesity and spine surgery was conducted using PubMed, Google Scholar, and Cochrane databases.

Results Obesity contributes to disk degeneration and low back pain and potentially increases the risk of developing operative pathology. Obese patients undergoing spine surgery have a higher risk of developing postoperative complications, particularly surgical site infection and venous thromboembolism. Though functional outcomes in this population may not mirror the general population, the treatment effect associated with surgery is at least equivalent if not better in obese individuals. This reduction is primarily due to worse outcomes associated with nonoperative treatment in the obese population.

Conclusion Obese individuals represent a unique patient population with respect to nonoperative treatment, postoperative complication rates, and functional outcomes. However, given the equivalent or greater treatment effect of surgery, this comorbidity should not prohibit obese patients from undergoing operative intervention. Future investigations in this area should attempt to develop strategies to minimize complications and improve outcomes in obese individuals and also examine the role of controlled weight loss preoperatively to mitigate these risks.

Keywords
- obesity
- surgical site infection
- lumbar spine
- venous thromboembolism

Introduction

Obesity represents a growing public health challenge, with an estimated prevalence of 34.9% of U.S. adults, or 78.6 million people.[1] On an economic level, Americans spend 147 billion dollars annually to combat the health-related medical problems attributed to obesity.[2] The World Health Organization (WHO) defines obesity by calculating the body mass index (BMI). This measure is derived from dividing weight in kilograms by height in meters squared. Individuals with a BMI between 25 and 29.9 kg/m^2 are classified as overweight. Those with a BMI between 30 and 40 kg/m^2 are obese, and those with BMIs >40 kg/m^2 are considered morbidly obese. Obesity is associated with a litany of medical comorbidities including diabetes mellitus, hypertension, osteoarthritis, obstructive sleep apnea, and depression.[3–5] Though the effects on overall health are well documented, a growing body of literature suggests that obesity may contribute to increased rates of disk degeneration, spinal arthritis, and low back pain.[3,6–8] As the incidence of obesity continues to rise in the general population, spine surgeons will undoubtedly perform more surgical procedures on these individuals. The purpose of this article

received
October 12, 2015
accepted
November 11, 2015
published online
January 15, 2016

DOI http://dx.doi.org/10.1055/s-0035-1570750.
ISSN 2192-5682.

is to detail the effects obesity has on the adult spine as well as the effects on postoperative complication rates and functional outcomes in individuals undergoing spine surgery through a review of the existing literature.

Effects on the Spinal Column

Obesity has been linked with increased rates of disk degeneration, low back pain, sciatica, and spine surgery.[9–13] In a review of magnetic resonance imaging of the spine in 975 individuals, Teraguchi et al reported a significantly higher incidence of disk degeneration in the cervical (odds ratio [OR]: 1.60; $p < 0.05$), thoracic (OR: 3.12; $p < 0.0001$), and lumbar spine (OR: 2.56; $p < 0.05$) in obese subjects.[11] In a separate meta-analyses, Shiri and colleagues reported a significant association between obesity and low back pain, sciatica, and surgery for disk herniation.[9,10] Although intuitively it seems that the most obvious cause of obesity-related disk degeneration would be the greater mechanical loads imparted on the spine in heavier individuals, more recent research suggests a multifactorial origin. Additional explanations for the increased incidence of disk degeneration in obese individuals include obesity-related chronic inflammation, gene–environment interaction, and decreased blood supply to the disk leading to ischemic changes.

The chronic inflammatory state associated with obesity likely plays a role in the development of disk degeneration. Adipocytes are the body's basic fat-building cells. Although serving as a reservoir for energy storage, adipocytes also have an endocrine function through the release of cytokines, termed *adipokines*. The most heavily researched of these adipokines is leptin. Higher levels of systemic adipokines have been associated the development and progression of osteoarthritis in multiple joints. In a study of patients undergoing knee arthroplasty and arthroscopy, Dumond et al noted that higher concentrations of leptin in arthritic joints correlated with BMI.[14] Furthermore, leptin has also been associated with the progression of osteoarthritis in the hand.[15] In the vertebral column, adipokines contribute to cell clustering and matrix degradation within the disk tissue.[12,16,17] These findings suggest that adipokines play a role in the development of joint degeneration in both the axial and appendicular skeleton.

A gene–environment interaction may also contribute to the increased incidence of disk degeneration in the obese population. An arginine-to-tryptophan change in the *COL9A3* gene (Trp 3allele) has been independently associated with disk degeneration and sciatica.[18,19] In a cross-sectional study of 135 middle-aged men, Solovieva et al reported a synergistic relationship between obesity and this Trp 3 allele for the development of decreased disk height, posterior disk bulge, and disk signal changes on magnetic resonance imaging.[18] This finding suggests that a genetic predisposition in a portion of the obese population may increase their risk of disk degenerative changes and potential back-related disability.

Obese individuals may experience higher rates of spine pathology secondary to atherosclerotic and lipid-related disk ischemia. The blood supply to the intraosseous portion of the vertebral body is derived from the nutrient and metaphyseal arteries.[20] Each of these has centrifugal branches that terminate at the end plates and arise from the lumbar arteries, which branch directly from the aorta.[20] Having no direct blood supply, the intervertebral disk relies on diffusion from the adjacent vertebral bodies as its only source of nutrition and to expel cellular waste such as lactate. A compromise in this supply of nutrients, oxygen and waste expulsion at any point along the vascular tree reduces cellular viability and proteoglycan production, which leads to disk degeneration.[21] In two postmortem studies, calcification of the abdominal aorta and atherosclerotic stenosis of the lumbar arteries were correlated with an increased risk of disk degeneration and low back pain.[22,23] Higher plasma levels of apolipoprotein E have also been associated with chronic lumbar pain.[24] As obesity has been linked with both apolipoprotein E and peripheral vascular disease,[25] the ischemia-related disk degeneration produced by these two risk factors may also contribute to the higher incidence of spine-related conditions in this population.

Though the exact contribution each of these factors plays in the development of spine-related pain is not fully understood, the existing evidence suggests a multifactorial problem. Developing a more complete understanding of this phenomenon may improve existing theories on the development of disk degeneration and help identify novel therapies aimed at prevention of disk degeneration and neurologic compression in this population.

Effects on Postoperative Complications

Obesity has been associated with longer operative times, increased blood loss, increased treatment cost, higher risk of mortality, and higher rates of both surgical site infection and venous thromboembolism.[6,26–36] The complex relationship between increased rates of postoperative complications and obesity is likely at least partially dependent on confounding variables within this population. Obese patients undergoing spine surgery have higher rates of medical comorbidities, such as diabetes mellitus and coronary arterial disease, which serve as independent risk factors for postoperative adverse events.[5,6,35,37] To this end, in a retrospective cohort study of 49,314 patients undergoing elective lumbar procedures, Seicean et al reported higher rates of both complications and readmission in individuals with BMI greater than 30 kg/m^2.[38] However, on matched propensity scoring controlling for medical comorbidities, only individuals with BMI $>$ 40 kg/m^2 demonstrated a significantly higher rate of complications, readmission, or nonroutine discharge.[38] The results of this study suggest that comorbid medical conditions contribute to the increased rate of postoperative complications. However, in the morbidly obese population, obesity alone is an independent risk factor for complications.

Multiple investigators have also documented longer operative times and increased blood loss in obese patients.[6,27,32,35,37,39] Each of these factors have been correlated with an increased incidence of postoperative complications.[30,40] Longer operative times contribute to higher complication rates by increasing the amount of soft tissue ischemia from retraction, exposing sterile instrumentation to a higher likelihood of contamination, and increasing

the total anesthetic and positioning time. Larger volumes of blood loss can lead to both higher rates of postoperative transfusion and hematoma formation, which have both been identified as risk factors for postoperative complications.[41–43]

The presence of obesity has been correlated with higher rates of postoperative infection in multiple investigations (►**Table 1**).[6,26,27,30,35,38,44,45] In a retrospective analysis of the American College of Surgeons National Surgeons Quality Improvement Program (NSQIP) database, Lim et al noted a significantly higher risk of surgical site infection in the obese population (OR: 1.628, 95% confidence interval [CI]: 1.042 to 2.544).[30] However, in a meta-analysis reviewing 24 studies, Jiang et al reported a significant relationship between obesity and surgical site infection (OR: 2.33, 95% CI 1.94 to 2.79).[27] Though each of these investigations associated risk of infection with obesity (BMI $>$ 30 kg/m^2), Mehta and colleagues suggested that the distribution of adipose tissue plays a significant role in the development of surgical site infection following spine surgery.[44,46] To this end, the author found significant associations between infection and the skin-to-lamina distance as well as the thickness of the subcutaneous tissue as measured on magnetic resonance imaging in cervical and lumbar procedures.[44,46] These studies introduce the idea that body habitus, rather than BMI, may be a better predictor of surgical site infection, which may be particularly true in more muscular individuals with relatively high BMIs but little adipose tissue. Understanding this potential limitation in the use of BMI alone may help practicing spine surgeons better understand this elevated risk of postoperative infection.

Another commonly feared complication associated with obesity following spine surgery is venous thromboembolism. In the general population, obesity is an independent risk factor for both deep venous thrombosis and pulmonary embolism.[47,48] This strong association is the result of (1) obesity-related chronic inflammation, (2) impairment of normal fibrinolysis, (3) increased thrombin generation and increased platelet activity.[49,50] A higher risk of venous thromboembolism has also been noted in obese individuals undergoing elective spine surgery by multiple investigators (►**Table 2**).[27,29,31] In a retrospective analysis of 24,196 patients undergoing lumbar spine surgery from the NSQIP database, Marquez-Lara reported significantly higher rates of thromboembolic events in individuals with a BMI greater than 25.[29] In a meta-analysis analyzing six studies, Jiang et al noted a significantly higher risk of venous thromboembolic complications in obese individuals undergoing spine surgery (OR: 3.15, 95% CI 1.92 to 5.17).[27] The increased incidence of this potentially fatal complication in obese patients should be considered in the formulation of a postoperative anticoagulation strategy, particularly those with mobilization precautions or comorbid conditions that would further predispose them to thromboembolic events.

Obesity is also widely believed to be a risk factor for perioperative peripheral nerve injury.[33,51,52] The causes of this phenomenon are likely related to higher compressive forces in contact areas and potentially longer operative times. To date, no large studies validating obesity as a risk for positional nerve injury in patients undergoing spine surgery have been performed. However, in a small retrospective case

Table 1 Incidence of infection in the obese population undergoing spine surgery

First author	Study design	Subjects (*n*)	Incidence of infection	Comments
De la Garza-Ramos[6]	Retrospective cohort study	732	BMI $<$ 29.9: 3.78%; BMI $>$ 30: 12.86% ($p = 0.001$)	Review of one- to three-level lumbar posterolateral fusions
Djurasovic[45]	Retrospective case series	270	BMI $<$ 29.9: 0.6%; BMI $>$ 30: 5.5% ($p = 0.018$)	Review of lumbar fusions for degenerative causes
Higgins[35]	Retrospective case series	801	BMI $<$ 29.9: 1.5%; BMI 30–40: 4.2% ($p = 0.03$); BMI $>$ 40: 15.0% ($p < 0.001$)	Study includes all instrumented cases at a single institution from all regions of the spine
Jiang[27]	Meta-analysis	93,183	OR BMI $>$ 30 kg/m^2: 2.33; 95% CI: 1.94–2.79	Evidence graded as moderate
Lim[30]	Retrospective multivariate analysis	3,353	OR BMI $>$ 30 kg/m^2: 1.63; 95% CI: 1.042–2.544 ($p = 0.032$)	Review of single-level lumbar fusion cases
Marquez-Lara[29]	Retrospective database review	24,196	BMI $<$ 24.9: 0.7%; BMI $>$ 25: 1.3% ($p < 0.001$)	Relative risk of infection increased as BMI increased (BMI $>$ 40: RR: 3.8, 95% CI: 2.5–5.9; $p = 0.001$)
Mehta[44]	Retrospective case series	298	BMI $<$ 29.9: 5.1%; BMI $>$ 30: 12.3% ($p = 0.025$)	Skin-to-lamina distance and the thickness of the subcutaneous tissue were also associated with increased rates of infection
Soroceanu[26]	Retrospective database review	241	OR BMI $>$ 30 kg/m^2: 4.88 ($p = 0.02$)	Review of adult spinal deformity cases

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio; RR, relative risk.

Table 2 Incidence of venous thromboembolism in the obese population undergoing spine surgery

First author	Study design	Subjects (*n*)	Incidence of infection	Comments
Jiang[27]	Meta-analysis	85,085	OR BMI > 30 kg/m^2: 3.15, 95% CI: 1.92–5.17	Evidence graded as moderate
Kalanithi[31]	Retrospective database review	84,607	OR BMI > 40 kg/m^2: PE/DVT: 3.34, 95% CI: 2.01–5.54	Review of morbidly obese patients undergoing cervical or lumbar fusion
Marquez-Lara[29]	Retrospective database review	24,196	RR for BMI > 25.0 kg/m^2: DVT: 2.0, 95% CI: 1.2–3.5 ($p = 0.009$); PE: 1.9, 95% CI: 1.0–3.6 ($p < 0.001$)	Reviewed results after all lumbar spine surgeries

Abbreviations: CI, confidence interval; DVT, deep vein thrombosis; OR, odds ratio; PE, pulmonary embolism; RR, relative risk.

series, Patel et al noted nerve compression injuries only in individuals with morbid obesity (BMI > 40 kg/m^2).[33] Though the small sample size combined with the low number of nerve injuries observed in this study ($n = 2$) prevent drawing definitive conclusions, extra care should be dedicated to the positioning of obese patients prior to spine surgery.

Effects on Functional Outcomes

Obesity's influence on clinical outcomes is controversial. In a retrospective review of 271 patients undergoing lumbar fusion 2 years after surgery, Djurasovic et al reported no significant difference in mean improvement seen between obese and nonobese patients in respect to Short Form-36 (SF-36) physical composite summary and Oswestry Disability Index (ODI) (ΔSF-36 physical composite summary: 4.22 versus 6.17, $p = 0.147$; ΔODI: 15.35 versus 14.03, $p = 0.602$).[45] Similarly, in a comparison of surgical outcomes in the obese and nonobese populations within the Spine Patient Outcomes Research Trial (SPORT) data, Rihn et al noted no significant difference in outcome measures at 4 years in both lumbar stenosis and degenerative spondylolisthesis, with the exception of SF-36 physical function scores in the degenerative spondylolisthesis population (22.1 versus 27.9, $p = 0.022$).[28] To the contrary, in a review of 2,633 patients from the Swedish Spine Registry, Knutsson et al noted that obese patients (BMI > 30 kg/m^2) had significantly worse ODI and EuroQol Group Index (EQ-5D) scores than normal weight patients (BMI < 25 kg/m^2; EQ-5D 0.56 versus 0.64, $p < 0.001$; ODI 33 versus 25, $p < 0.001$) and less mean improvement 2 years after surgery.[53] In this study, the obese population also experienced significantly greater risk for dissatisfaction with the results of surgery (OR: 1.73, 95% CI: 1.36 to 2.19).[53] In a review of patients undergoing surgery for lumbar disk herniation from the SPORT data, Rihn et al noted significantly less improvement in ODI and all components of the SF-36 scores as well as the Sciatica Bothersomeness and Low Back Pain Bothersomeness Indices in the obese population.[54]

Although these investigations provide contradictory information on whether obese patients experience less improvement with surgery, they should be interpreted carefully as obesity has been associated with worse baseline functional scores and less improvement with nonoperative treatment.[26,28,37,45,53,54] Differences in baseline functional measures were demonstrated in the investigation by Knutsson et al, where obese individuals had worse preoperative EQ-5D and ODI scores than the normal weight population (EQ-5D: 0.34 versus 0.38, $p = 0.0026$; ODI: 46 versus 42, $p < 0.0001$).[53] Djurasovic et al also noted higher (worse) pretreatment ODI scores in the obese population (ODI: 55.83 versus 51.42, $p = 0.017$).[45] The obese population also seems to receive less benefit from nonoperative treatment. In multiple examinations of the SPORT data, obese individuals with lumbar disk herniation, lumbar stenosis, or degenerative spondylolisthesis have demonstrated less improvement in functional outcome measures at up to 4-year follow-up.[28,37,54] These differences in both baseline disability and response to nonoperative treatment limit the value of directly comparing long-term functional outcome measures in the obese and nonobese populations to determine the efficacy of surgery.

In this context, it is more important to consider the treatment effect of operative intervention as a measure of therapeutic success. The treatment effect is defined as the difference between mean improvement in a surgical population compared with mean improvement associated with nonoperative treatment in a similar population. When considering this measure of success, obese patients have similar or better responses to operative intervention than their nonobese counterparts.[28,37,54] In examining this metric, the SPORT data likely provides the most complete insight due to the fact it represents a large prospective data set collected at multiple institutions (►**Tables 3**, **4**, and **5**). In a subgroup analysis of these results, Rihn et al reported equivalent surgical treatment effects in the obese and nonobese populations for lumbar disk herniation using SF-36 and ODI scores.[54] However, obese individuals had greater treatment effects with surgery in both the lumbar stenosis (SF-36 physical profile) and degenerative spondylolisthesis (ODI) populations.[28] The authors attributed these differences in treatment effect to the worse outcomes observed with nonoperative management in the obese group. Though controversy may persist on whether obese patients experience the same magnitude of functional improvement after surgery, the greater treatment effects noted indicate that surgery,

Table 3 Treatment effect of surgery for lumbar stenosis[28]

Outcome	1 y	4 y
SF-36 bodily pain		
BMI < 30 kg/m^2	14.6	12.6
BMI > 30 kg/m^2	17.2	15.9
p	0.47	0.44
SF-36 physical function		
BMI < 30 kg/m^2	13.5	7.4
BMI > 30 kg/m^2	16.3	12.8
p	0.40	0.17
ODI		
BMI < 30 kg/m^2	−10.1	−7.4
BMI > 30 kg/m^2	−15.7	−13.9
p	0.036	0.037

Abbreviations: BMI, body mass index; ODI, Oswestry Disability Score; SF-36, Short Form-36.

Table 4 Treatment effect of surgery for lumbar spondylolisthesis[28]

Outcome	1 y	4 y
SF-36 bodily pain		
BMI < 30 kg/m^2	16.7	13.8
BMI > 30 kg/m^2	20.7	17.2
p	0.26	0.43
SF-36 physical function		
BMI < 30 kg/m^2	16	14
BMI > 30 kg/m^2	20.7	25.6
p	0.17	0.004
ODI		
BMI < 30 kg/m^2	−15.4	−12.6
BMI > 30 kg/m^2	−19.6	−17.5
p	0.11	0.12

Abbreviations: BMI, body mass index; ODI, Oswestry Disability Score; SF-36, Short Form-36.

compared with nonoperative treatment, is at least as advantageous in this group of patients.

Conclusion

With important differences in postoperative complication rates and response to both operative and nonoperative treatment, obese individuals represent a unique patient population. As the global incidence of obesity continues to rise, obese patients will likely constitute a larger portion of the spine surgeon's practice. Though the current literature points to increased rates of postoperative complications, particularly infection and venous thromboembolic events, there is also a suggestion of potentially greater treatment effects with surgical intervention owing largely to worse outcomes with nonoperative treatment. Future research in this field should examine measures to minimize the complication rates in this population as well as the effect of controlled weight loss before surgery on complications and outcomes.

Table 5 Treatment effect of surgery for lumbar disk herniation[54]

Outcome	1 y	4 y
SF-36 bodily pain		
BMI < 30 kg/m^2	13.4	16.5
BMI > 30 kg/m^2	17.5	13.3
p	0.18	0.35
SF-36 physical function		
BMI < 30 kg/m^2	14.8	15.7
BMI > 30 kg/m^2	19	14.3
p	0.13	0.64
ODI		
BMI < 30 kg/m^2	−13.3	−14
BMI > 30 kg/m^2	−18.5	−12.3
p	0.021	0.50

Abbreviations: BMI, body mass index; ODI, Oswestry Disability Score; SF-36, Short Form-36.

Disclosures
Keith L. Jackson II: none
John G. Devine: none

Disclaimer
The views expressed herein are those of the author(s) and do not reflect the official policy of the Department of the Army, Department of Defense, or the U.S. Government.

References

1 Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA 2014; 311(8):806–814
2 Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer-and service-specific estimates. Health Aff (Millwood) 2009;28(5):w822–w831
3 Liuke M, Solovieva S, Lamminen A, et al. Disc degeneration of the lumbar spine in relation to overweight. Int J Obes 2005;29(8): 903–908
4 Marcus DA. Obesity and the impact of chronic pain. Clin J Pain 2004;20(3):186–191
5 Pi-Sunyer X. The medical risks of obesity. Postgrad Med 2009; 121(6):21–33
6 De la Garza-Ramos R, Bydon M, Abt NB, et al. The impact of obesity on short- and long-term outcomes after lumbar fusion. Spine (Phila Pa 1976) 2015;40(1):56–61

7 Gandhi R, Woo KM, Zywiel MG, Rampersaud YR. Metabolic syndrome increases the prevalence of spine osteoarthritis. Orthop Surg 2014;6(1):23–27
8 Romero-Vargas S, Zárate-Kalfópulos B, Otero-Cámara E, et al. The impact of body mass index and central obesity on the spinopelvic parameters: a correlation study. Eur Spine J 2013;22(4): 878–882
9 Shiri R, Solovieva S, Husgafvel-Pursiainen K, et al. The role of obesity and physical activity in non-specific and radiating low back pain: the Young Finns study. Semin Arthritis Rheum 2013; 42(6):640–650
10 Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between obesity and low back pain: a meta-analysis. Am J Epidemiol 2010;171(2):135–154
11 Teraguchi M, Yoshimura N, Hashizume H, et al. Prevalence and distribution of intervertebral disc degeneration over the entire spine in a population-based cohort: the Wakayama Spine Study. Osteoarthritis Cartilage 2014;22(1):104–110
12 Samartzis D, Karppinen J, Cheung JP, Lotz J. Disk degeneration and low back pain: are they fat-related conditions? Global Spine J 2013;3(3):133–144
13 Samartzis D, Karppinen J, Mok F, Fong DY, Luk KD, Cheung KM. A population-based study of juvenile disc degeneration and its association with overweight and obesity, low back pain, and diminished functional status. J Bone Joint Surg Am 2011;93(7): 662–670
14 Dumond H, Presle N, Terlain B, et al. Evidence for a key role of leptin in osteoarthritis. Arthritis Rheum 2003;48(11):3118–3129
15 Yusuf E, Ioan-Facsinay A, Bijsterbosch J, et al. Association between leptin, adiponectin and resistin and long-term progression of hand osteoarthritis. Ann Rheum Dis 2011; 70(7):1282–1284
16 Konttinen YT, Kääpä E, Hukkanen M, et al. Cathepsin G in degenerating and healthy discal tissue. Clin Exp Rheumatol 1999;17(2):197–204
17 Zhao CQ, Liu D, Li H, Jiang LS, Dai LY. Expression of leptin and its functional receptor on disc cells: contribution to cell proliferation. Spine (Phila Pa 1976) 2008;33(23):E858–E864
18 Solovieva S, Lohiniva J, Leino-Arjas P, et al. COL9A3 gene polymorphism and obesity in intervertebral disc degeneration of the lumbar spine: evidence of gene-environment interaction. Spine (Phila Pa 1976) 2002;27(23):2691–2696
19 Solovieva S, Lohiniva J, Leino-Arjas P, et al. Intervertebral disc degeneration in relation to the COL9A3 and the IL-1ss gene polymorphisms. Eur Spine J 2006;15(5):613–619
20 Ratcliffe JF. The arterial anatomy of the adult human lumbar vertebral body: a microarteriographic study. J Anat 1980;131(Pt 1):57–79
21 Horner HA, Urban JP. 2001 Volvo Award Winner in Basic Science Studies: Effect of nutrient supply on the viability of cells from the nucleus pulposus of the intervertebral disc. Spine (Phila Pa 1976) 2001;26(23):2543–2549
22 Kauppila LI, Tallroth K. Postmortem angiographic findings for arteries supplying the lumbar spine: their relationship to low-back symptoms. J Spinal Disord 1993;6(2):124–129
23 Kauppila LI. Prevalence of stenotic changes in arteries supplying the lumbar spine. A postmortem angiographic study on 140 subjects. Ann Rheum Dis 1997;56(10):591–595
24 Cameron BM, VanderPutten DM, Merril CR. Preliminary study of an increase of a plasma apolipoprotein E variant associated with peripheral nerve damage. A finding in patients with chronic spinal pain. Spine (Phila Pa 1976) 1995;20(5):581–589, discussion 589–590
25 Karagiannides I, Abdou R, Tzortzopoulou A, Voshol PJ, Kypreos KE. Apolipoprotein E predisposes to obesity and related metabolic dysfunctions in mice. FEBS J 2008;275(19):4796–4809
26 Soroceanu A, Burton DC, Diebo BG, et al; International Spine Study Group. Impact of obesity on complications, infection, and patient-reported outcomes in adult spinal deformity surgery. J Neurosurg Spine 2015; July 31 (Epub ahead of print)
27 Jiang J, Teng Y, Fan Z, Khan S, Xia Y. Does obesity affect the surgical outcome and complication rates of spinal surgery? A meta-analysis. Clin Orthop Relat Res 2014;472(3):968–975
28 Rihn JA, Radcliff K, Hilibrand AS, et al. Does obesity affect outcomes of treatment for lumbar stenosis and degenerative spondylolisthesis? Analysis of the Spine Patient Outcomes Research Trial (SPORT). Spine (Phila Pa 1976) 2012;37(23): 1933–1946
29 Marquez-Lara A, Nandyala SV, Sankaranarayanan S, Noureldin M, Singh K. Body mass index as a predictor of complications and mortality after lumbar spine surgery. Spine (Phila Pa 1976) 2014; 39(10):798–804
30 Lim S, Edelstein AI, Patel AA, Kim BD, Kim JY. Risk factors for postoperative infections following single level lumbar fusion surgery. Spine (Phila Pa 1976) 2014
31 Kalanithi PA, Arrigo R, Boakye M. Morbid obesity increases cost and complication rates in spinal arthrodesis. Spine (Phila Pa 1976) 2012;37(11):982–988
32 Buerba RA, Fu MC, Gruskay JA, Long WD III, Grauer JN. Obese class III patients at significantly greater risk of multiple complications after lumbar surgery: an analysis of 10,387 patients in the ACS NSQIP database. Spine J 2014;14(9):2008–2018
33 Patel N, Bagan B, Vadera S, et al. Obesity and spine surgery: relation to perioperative complications. J Neurosurg Spine 2007;6(4): 291–297
34 Schuster JM, Rechtine G, Norvell DC, Dettori JR. The influence of perioperative risk factors and therapeutic interventions on infection rates after spine surgery: a systematic review. Spine (Phila Pa 1976) 2010;35(9, Suppl):S125–S137
35 Higgins DM, Mallory GW, Planchard R, et al. Understanding the impact of obesity on short-term outcomes and in-hospital costs after instrumented spinal fusion. Neurosurgery 2016;78(1):127–132
36 Minhas SV, Chow I, Jenkins TJ, Dhingra B, Patel AA. Preoperative predictors of increased hospital costs in elective anterior cervical fusions: a single-institution analysis of 1,082 patients. Spine J 2015;15(5):841–848
37 McGuire KJ, Khaleel MA, Rihn JA, Lurie JD, Zhao W, Weinstein JN. The effect of high obesity on outcomes of treatment for lumbar spinal conditions: subgroup analysis of the spine patient outcomes research trial. Spine (Phila Pa 1976) 2014;39(23): 1975–1980
38 Seicean A, Alan N, Seicean S, et al. Impact of increased body mass index on outcomes of elective spinal surgery. Spine (Phila Pa 1976) 2014;39(18):1520–1530
39 Hardesty CK, Poe-Kochert C, Son-Hing JP, Thompson GH. Obesity negatively affects spinal surgery in idiopathic scoliosis. Clin Orthop Relat Res 2013;471(4):1230–1235
40 Wimmer C, Gluch H, Franzreb M, Ogon M. Predisposing factors for infection in spine surgery: a survey of 850 spinal procedures. J Spinal Disord 1998;11(2):124–128
41 Perry JW, Montgomerie JZ, Swank S, Gilmore DS, Maeder K. Wound infections following spinal fusion with posterior segmental spinal instrumentation. Clin Infect Dis 1997;24(4): 558–561
42 Schwarzkopf R, Chung C, Park JJ, Walsh M, Spivak JM, Steiger D. Effects of perioperative blood product use on surgical site infection following thoracic and lumbar spinal surgery. Spine (Phila Pa 1976) 2010;35(3):340–346
43 Woods BI, Rosario BL, Chen A, et al. The association between perioperative allogeneic transfusion volume and postoperative infection in patients following lumbar spine surgery. J Bone Joint Surg Am 2013;95(23):2105–2110
44 Mehta AI, Babu R, Karikari IO, et al. 2012 Young Investigator Award winner: The distribution of body mass as a significant risk factor for lumbar spinal fusion postoperative infections. Spine (Phila Pa 1976) 2012;37(19):1652–1656

45 Djurasovic M, Bratcher KR, Glassman SD, Dimar JR, Carreon LY. The effect of obesity on clinical outcomes after lumbar fusion. Spine (Phila Pa 1976) 2008;33(16):1789–1792
46 Mehta AI, Babu R, Sharma R, et al. Thickness of subcutaneous fat as a risk factor for infection in cervical spine fusion surgery. J Bone Joint Surg Am 2013;95(4):323–328
47 Stein PD, Beemath A, Olson RE. Obesity as a risk factor in venous thromboembolism. Am J Med 2005;118(9): 978–980
48 Parkin L, Sweetland S, Balkwill A, Green J, Reeves G, Beral V; Million Women Study Collaborators. Body mass index, surgery, and risk of venous thromboembolism in middle-aged women: a cohort study. Circulation 2012;125(15):1897–1904
49 Blokhin IO, Lentz SR. Mechanisms of thrombosis in obesity. Curr Opin Hematol 2013;20(5):437–444
50 Samad F, Ruf W. Inflammation, obesity, and thrombosis. Blood 2013;122(20):3415–3422
51 American Society of Anesthesiologists Task Force on Prevention of Perioperative Peripheral Neuropathies. Practice advisory for the prevention of perioperative peripheral neuropathies: an updated report by the American Society of Anesthesiologists Task Force on prevention of perioperative peripheral neuropathies. Anesthesiology 2011;114(4):741–754
52 Warner MA, Warner ME, Martin JT. Ulnar neuropathy. Incidence, outcome, and risk factors in sedated or anesthetized patients. Anesthesiology 1994;81(6):1332–1340
53 Knutsson B, Michaëlsson K, Sandén B. Obesity is associated with inferior results after surgery for lumbar spinal stenosis: a study of 2633 patients from the Swedish spine register. Spine (Phila Pa 1976) 2013;38(5):435–441
54 Rihn JA, Kurd M, Hilibrand AS, et al. The influence of obesity on the outcome of treatment of lumbar disc herniation: analysis of the Spine Patient Outcomes Research Trial (SPORT). J Bone Joint Surg Am 2013;95(1):1–8